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## Electrical Stimulation Decreases Neuralgic Pain after Trigeminal Deafferentation

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Running head: TENS for trigeminal anaesthesia dolorosa

## INTRODUCTION

Trigeminal nerve activity mediates head pain after cranial injuries, and also contributes to idiopathic syndromes such as cluster headache and trigeminal neuralgia. Nevertheless, chronic facial pain sometimes develops after lesion of the trigeminal nerve. Here we report on a patient who developed dysaesthesia and stabbing pain in her face after the trigeminal ganglion was destroyed. Transcutaneous electrical stimulation within the distribution of trigeminal deafferentation alleviated the anaesthesia dolorosa and neuralgic jabs, suggesting that sensory afferents that bypassed the trigeminal ganglion mediated therapeutic effects.

## CASE REPORT

*History and presentation.* Ten years before the current investigation, a 39 year-old woman developed left facial numbness, decreased left eye abduction with diplopia on extreme left gaze, and mild left-sided conduction hearing loss. Subsequently, a sphenoid meningioma involving the central skull base that extended through the left cavernous sinus was identified, and the intracranial portion was removed along with most of the left trigeminal ganglion. After the surgical procedure, the left side of the patient's face was numb apart from minor sensation in the chin. However, most ocular and facial movements remained intact and artificial tears were not required. She also had a persistently small left pupil and partial left-sided ptosis, and the left side of her forehead did not sweat during body heating or when she exercised. Nine years after the meningioma was removed, an MRI scan of the central skull base indicated residual

tumour in the left sphenoid bone, cavernous sinus, the optic canal and Meckel's cave that did not require further surgery or radiation treatment.

Several months after the meningioma was removed, left-sided jabs of pain developed in all three divisions of the trigeminal nerve. The pain initially responded to carbamazepine, sodium valproate and amitriptyline, but these drugs were discontinued because of intolerable side effects. The pain continued over the next ten years and at the time of investigation was described as a tic-like stabbing sensation above the left upper lip, in the left side of the nose, the cheek or along an indented scar in the left forehead where bone had been removed surgically. The jabs began as pins and needles that quickly intensified into 5-10 painful stabs over the next ten seconds. Stabbing pain recurred for approximately ten minutes every hour without any immediate identifiable trigger, but appeared to be aggravated by stress. The stabbing pain was superimposed on a constant dysaesthesia deep in the left cheek, jaw and behind the left eye, but neither the neuralgic jabs nor the dysaesthesia were accompanied by lacrimation, conjunctival injection or other autonomic disturbances. At low intensities the dysaesthesia felt like tiny insects crawling around inside her face or like local anaesthesia wearing off. At higher intensities the dysaesthesia developed into a dull pain, rather like a mild toothache, that moved continuously from one site to another.

*Investigations.* The patient provided informed consent for the investigations, which were approved by the Murdoch University Ethics Committee. Sensory testing with thin nylon hairs indicated complete loss of light tactile sensation from the forehead to the chin on the left side, whereas deep pressure stimulation with firm bristles or an algometer evoked a dull painless sensation at each site that began at higher pressures than on the

right. The patient could not detect 4°C stimuli in the left forehead or cheek, but could detect slight coolness in the chin. She could not detect warmth or heat pain in the left forehead, cheek or chin when the skin was heated at 0.5°C/s from 32-49°C with a radiant heat lamp. To investigate the effect of counter-irritation on facial pain, the patient immersed her right hand in 10°C water for one minute. Hand pain was rated as 9 (extremely painful) on a 0-10 scale of pain intensity whereas facial dysaesthesia decreased from 2 (mild pain) to 0 for 1-2 minutes. The dysaesthesia gradually returned to the previous intensity over the next ten minutes. Effects were similar after the patient immersed her left hand in the cold water.

On another occasion, concentric electrodes were attached to the supraorbital region on each side of the forehead, to stimulate intradermal trigeminal nociceptive afferents (1). Blink reflexes were recorded bilaterally from surface electrodes attached below the lower eyelids and 2-3 cm laterally. Current intensity (monopolar square wave pulses, 0.3 msec duration, interstimulus interval greater than 15 s) was increased in 0.1 to 0.3 mA steps to identify the pain and blink reflex thresholds, first on the right side and then the left. On the right side of the forehead, 0.7 mA stimuli consistently evoked mild pain, and bilateral blink reflexes began around 1.2 mA (indicating that both facial nerves were intact) (Figure 1). In contrast, the patient was unaware of any sensation on the left side of the forehead for stimuli up to 27 mA (the maximum intensity employed), and blink reflexes were absent (indicating that the trigeminal nerve was lesioned). At the start of the session, the left-sided stabbing pain and dysaesthesia was rated as 4.5 on the 0-10 scale of pain intensity, and remained unchanged when the right side of the forehead was stimulated with electric currents. However, pain decreased to 1 (slight pain) after left-

sided stimulation, even though the patient did not detect any of the electrical stimuli. Pain was minimal for several hours afterwards.

*Management.* To determine whether the decrease in facial pain after electrical stimulation could be attributed to a placebo response, real or sham stimuli were applied to the left side of the forehead on different occasions. Dysaesthesia decreased from moderate (4/10) to slight (1/10) after 2-20 mA stimulation from the concentric electrode (intermittent 0.3 msec monopolar square wave pulses), and remained at 0-1 over the next six hours. In addition, the neuralgic jabs recurred infrequently. However, during and after sham stimulation dysaesthesia decreased only marginally, from 2.5 to 1.5, and had increased to a rating of 3 six hours later. Neuralgic jabs decreased for an hour after sham stimulation but then recurred at the previous intensity. Pain was inhibited over a period of several months when the patient self-administered transcutaneous electrical nerve stimulation on the left side of her forehead and cheek every 1-2 days for 30-60 minutes (2 Hz at a current intensity that induced paraesthesiae when the electrode was applied to her forearm). The dysaesthesia and neuralgic jabs returned on several occasions when stimulation was discontinued for more than two days.

## **DISCUSSION**

Our patient developed neuralgic jabs and anaesthesia dolorosa several months after the left trigeminal ganglion was destroyed when a sphenoid meningioma was removed surgically. Although the origin of the neuralgic jabs is unclear, one possibility is that the residual meningioma compressed sensory afferents that bypassed the lesioned trigeminal ganglion, thereby triggering abnormal spike discharge (2). The mechanism of anaesthesia dolorosa is also uncertain, but could entail spontaneous activity in sensitized

central pain pathways in the absence of normal afferent input (3,4). A similar mechanism, possibly involving sensitization or disinhibition of pain pathways in the cingulate cortex, may contribute to chronic facial pain in patients with atypical trigeminal neuralgia (5). Electrical stimulation of the trigeminal ganglion and nerve root sometimes alleviates dysaesthesia in patients with residual facial sensation (6,7), suggesting that afferent input inhibits the spontaneous central discharge. In the present case, nociceptive blink reflexes and most sensory modalities were lost on the affected side of the face after removal of the trigeminal ganglion. Nevertheless, electrical stimulation of the affected forehead and cheek alleviated dysaesthesia and suppressed neuralgic jabs.

Although most facial sensations are conveyed to the central nervous system by the trigeminal nerves, certain sensations persist after section of the trigeminal sensory nerve root (8-12). For example, Spiller (12) noted that sensitivity to deep pressure persisted in patients who lost other modalities of facial sensation when the trigeminal ganglion was infiltrated by an intracranial tumour or the ganglion was removed surgically. Pressure, static two-point discrimination and vibration thresholds are higher on the affected than unaffected side in patients with unilateral lower motoneuron facial nerve palsy (13), suggesting that the facial nerve distributes sensory fibres to facial tissues (most likely pressure sensors and proprioceptors in muscle) (8-10). Helson (11) noted that sensitivity to deep pressure generally began to return within a month of sectioning the second and third divisions of the trigeminal nerve root, despite permanent loss of light tactile sensations. In addition, an extremely hot stimulus, between 60°C and 75°C, elicited a stinging or pricking sensation when applied to affected malar or oral regions if the stimulus was applied long enough for heat to penetrate below the surface. Remarkably,

this capacity was lost in patients who had also undergone a thoracic sympathectomy, although sensitivity to pressure remained intact.

The presence of Horner's syndrome and loss of facial sweating in our patient indicates that the cervical sympathetic pathway had been compromised, possibly during the surgical procedure or due to compression of the internal carotid plexus in the cavernous sinus by the meningioma. However, the facial nerve appeared to be intact because facial movements, lacrimation, and blinks to contralateral facial stimulation were preserved. Thus, the facial nerve might have provided an afferent pathway for deep pressure sensations (8,14). In cats, afferent fibres in the facial nerve project to the spinal trigeminal nucleus (15), thereby offering an ancillary pathway to the nucleus that might increase in prominence in the absence of normal trigeminal input.

Electrical stimulation of the trigeminal ganglion sometimes alleviates neuropathic pain in patients with residual facial sensation but is less effective for patients with marked sensory loss (6,7). Nevertheless, in the present case stimulating the affected side of the face was beneficial despite virtually complete loss of facial sensation apart from deep pressure. Moreover, electrical stimulation of trigeminal cutaneous nociceptive afferents failed to induce blink reflexes, even at stimulus intensities that would also be expected to excite intradermal non-nociceptive trigeminal afferents. Taken together, these findings suggest that excitation of sensory afferents that entered the brainstem via a pathway that bypassed the lesioned trigeminal ganglion (e.g., via pressure sensors or proprioceptors in the facial nerve) suppressed abnormal discharge within deafferented central trigeminal pathways. In favour of a direct inhibitory process, sham stimulation evoked only transient decreases in pain (perhaps due to a placebo effect). Immersion of the hand in painfully-



cold water also inhibited facial pain, consistent with activation of diffuse noxious inhibitory controls (16). However, this mechanism does not account for the therapeutic effect of electrical stimulation, because stimuli applied to the contralateral forehead were ineffective.

Curiously, electrical stimuli that inhibited facial dysaesthesia produced no sensations. These findings suggest that excitation of a sparse population of intradermal non-trigeminal sensory afferents, below the threshold of sensory perception, suppressed activity in central trigeminal neurons or higher-order pain pathways (5). Even more surprisingly, this inhibitory effect persisted for several hours, implying that afferent input somehow “reset” normal inhibitory influences on spontaneous discharge in these pain pathways. Similarly, stimulation of the motor cortex is beneficial for patients with trigeminal neuropathic pain, with periods of pain relief sometimes lasting several hours (17). The mechanism of this persistent analgesia is unknown.

In conclusion, our findings suggest that pressure sensations in a patient with neuralgic jabs and anaesthesia dolorosa following trigeminal deafferentation were mediated by sensory afferents that bypassed the lesioned trigeminal ganglion. Subliminal excitation of these fibres may also have mediated the therapeutic effect of electrical stimulation. Whether sensory afferents in the facial nerve generally play a role in trigeminal deafferentation syndromes requires further investigation.

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### FIGURE LEGEND

**Figure 1.** Blink reflexes to a 2.3 mA stimulus delivered from a concentric electrode attached to the right or left supraorbital region. Signals were rectified and filtered to remove electrical noise and baseline drift. Weak stimuli delivered from concentric electrodes generally evoke responses beginning approximately 40 msec after stimulus onset (corresponding to the nociceptive R2 component of the blink reflex) but do not evoke R1, indicating that the current excites cutaneous nociceptors but not deeper A $\beta$  fibres (1). Note that right-sided stimulation evoked bilateral blink reflexes, whereas left-sided stimulation was ineffective.

